A METHOD OF STUDYING THE AORTA BY DETERMINING THE SPEED OF PROPAGATION OF PULSE WAVES

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ABSTRACT

A method using simple equipment is presented for determining pulse wave velocity from pulse curve lag behind EKG. Pulse curve recording improves and pulse wave propagation time in the proximal segment of the aorta can be estimated. Comparison of propagation times reveals an atherosclerotic pathognomic ratio. Age changes have little and functional changes have no effect on the ratio. author

Among a number of instrumental methods for diagosing /78* atherosclerosis, the most promising (A. L. Myasnikov) is determination of the speed of propagation of the pulse wave through the vessels. As is well known, the thicker the vessel wall, the more rapidly the pulse wave travels along it. Normally the velocity of the aortal wave, based on the data of a number of authors (V. P. Nikitin, Yu. T. Pushkar') falls in the range of 5 to 10 m/sec, depending on age. In atherosclerosis, as a result of lipoid infiltration and

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*Numbers given in the margin indicate the pagination in the original foreign text.

the consequent sclerosis, the elasticity of the vessel walls increases, causing a corresponding increase in velocity to 15 to 25 m/sec.

However, the velocity of the pulse wave also increases in a number of other conditions: with increased intra-arterial pressure, increased tonic tension, and with age-related changes in the vessel walls. The investigator is always faced with the problem of differentiating these conditions from atherosclerosis. For this purpose we propose to compare the pulse wave propagation time at the proximal end of the aorta with the propagation time at the distal end, establishing between them the ratio $\frac{t_1:t}{t_1}$. This relationship will be different for atherosclerosis and the other conditions mentioned previously.

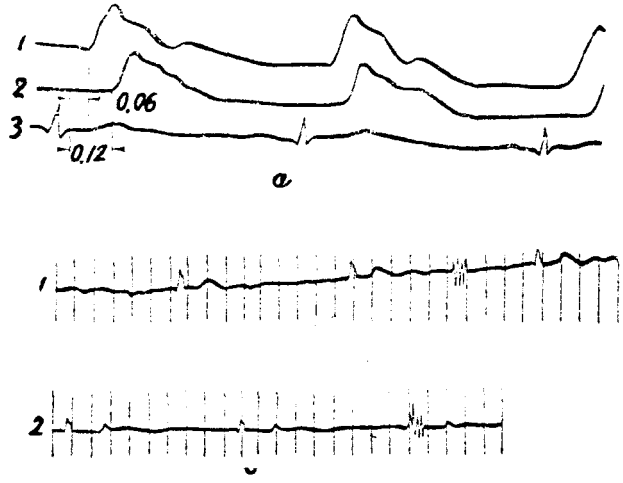
First of all it is necessary to consider the segments of path for which total elapsed time t and velocity V along a vessel of elastic type are determined. As is well known, in physics velocity is expressed as the distance traversed by a body (in our case, a pulse wave) per unit time, as shown in the formula $V = S/t$. If we place two pulse sensors, one at the beginning, the other at the end, of an artery, we can then determine the time t required for a pulse wave to travel from one pickup to the other (by the delay in the peripheral pulse with relation to the central pulse). By measuring the length of the artery S between the two pickups and dividing it by the time t, we find the distance travelled by the wave per unit time, i.e. the velocity V.

The path travelled by pulse waves in man is more complex. For one thing, pulse sensors cannot be placed directly on the aorta because of its buried anatomical position. They must be placed on branches of the aorta: the carotid and femoral arteries, from which the central and peripheral pulses, respectively, are recorded. The pulse wave, which is generated by contraction of the left ventricle, travels along the proximal sector of the aorta until it reaches the

branching off of the common carotid artery. At this point the wave splits in two, as it were, travelling upward along the carotid artery and downward along the continuation of the aorta. At the moment the pulse wave is recorded on the carotid artery, the pulse wave in the aorta has approximately reached the beginning of the descending portion of the thoracic aorta.

Thus, the first point, from which the pulse wave is timed in the aorta, corresponds to the beginning of the descending portion of the thoracic aorta, and the second point, to the beginning of the femoral artery, where the second pulse pickup is located. The area under study in this case will be that part of the arterial system which includes the beginning of the thoracic and the abdominal portions of the aorta and the iliac arteries, i.e., just those parts which according to the data of anatomical pathology are most subject to damage by the atherosclerotic process. For comparison purposes it is also important to determine the rate of propagation of the pulse wave along the proximal segment of the aorta.

Technically, this may be determined in two ways. The first and classical method is that of simultaneous recording on an oscillograph tape of two pulse curves. From the lag in the peripheral pulse curve (taken from the femoral artery) behind the central pulse curve (taken from the carotid) t is determined. The second method for determining t consists in the following: If /79 simultaneously with the two pulse curves from the carotid and femoral arteries a third oscillograph channel is used to record the EKG (see fig a), then t can be determined twice: classically, by the lag of the peripheral pulse behind the central, and by the difference in the lag of the peripheral and of the central pulse behind the EKG. This is done by determining for a number of



Methods of determining pulse wave propagation velocity
a - simultaneous recording of EKG (3) and carotid (1) and femoral (2) pulse curves; b - recording of sphygmograms from the carotid (1) and femoral (2) arteries on a single EKG channel.

points on the tape the time by which the carotid artery pulse curve (t_1) and femoral artery pulse curve (t_2) lag behind a given point on the EKG. The difference between t_2 and t_1 is the total elapsed time t.

As is evident from the above, the differences between these two methods are not essential, but merely procedural. At the same time this apparently at first glance simple supplementing of the pulse curves with an EKG trace lays the way open to new possibilities. First of all, the necessity of recording two pulse curves simultaneously is eliminated. Now the process can be done in two steps: first, record the carotid artery pulse curve on the tape simultaneously with the EKG, and determine t_1, and then use the same pickup to record the femoral artery pulse curve simultaneously with the EKG and determine t_2. And from this it is easy to pass on to a still simpler variant: with an ordinary one channel electrocardiograph record the EKG and

PULSE WAVE PROPAGATION TIME FOR THE PROXIMAL (t_1) AND DISTAL (t) SEGMENTS OF THE AORTA IN "HEALTHY" PERSONS, PATIENTS WITH HIGH BLOOD PRESSURE, AND PATIENTS WITH CORONARY-CARDIAC SCLEROSIS (NUMBER OF SUBJECTS SHOWN IN COLUMNS)

Time (in sec)	Healthy individuals		Patients with high Arterial Blood Pressure		Patients with coronary cardiac sclerosis	
	t_1	t	t_1	t	t_1	t
0.1		2		1	6	
0.09					3	
0.08	3	8	5	3	18	
0.07	8	6	5	13	21	
0.06	10	13	7	10	23	3
0.05	5	3	8	5	12	20
0.04	5	2	3	1	1	33
0.03	3		4			21
0.02						7
0.01						

a sphygmogram first from the carotid, then from the femoral, artery. It was this recording method which we used (see fig. b). Its advantage lies in the following: a) it permits elimination of the usual equipment, which makes the method feasible for wide use; b) two-step recording is technically simple, permitting full attention to be given to the registration of the pulse from each artery separately. This improves the quality of the pulse curve recording and makes it easy more exactly to determine the "points of coincidence" on the recording; and c) it becomes possible to study, not only the distal, but also the proximal aorta. This last possibility seems to us particularly important.

As already stated, with two-step recording the pulse curve lag reading may be from any starting point on the EKG. We started our reading, in accordance with the suggestions of F. M. Lebedev and Yu. S. Shternshis, from the end of the S peak (or if it were indistinct, from the base of the R peak), since,

in the opinion of those authors, this point coincides with the moment of generation of the pulse wave in the aorta. This last point is open to dispute.

If we accept the conclusion of a number of authors that the electrical and mechnical systole coincide in time, then the beginning of the EKG ventricular QRS complex actually corresponds in time (0.03 to 0.09 sec) to the tension phase; after it (after the S peak) comes the expulsion phase and the resulting generation of the pulse wave in the aorta.

However, the majority of authors at the present time hold the opinion that the mechnical systole commences a little bit after the electrical systole. In this case the point at the end of the S peak on the EKG may be taken to correspond approximately to the beginning of the pulse wave in the aorta, and the value t_1 to be approximately equal to the time required for the pulse wave to travel from the beginning of the aorta to the carotid artery, since it includes the time lag of the end of the tension phase of the mechanical systole behind the end of the tension phase of the electrical systole.

But the value t_1, as already stated, is arbitrarily taken as equal /80 to the propagation time of the pulse wave from the proximal segment of the aorta to a point located where the arch becomes the initial segment of the thoracic aorta. Beyond this point according to the conditions set by the method, recording begins for time t. In other words it may be said that t_1 approximately corresponds to the propagation time of the wave along the proximal segment of the aorta, while t corresponds to the propagation time along the distal segment. The use of t for deriving V for the distal segment of the aorta is well known. The question arises as to the use of t_1 for evaluating the elasticity of the proximal segment. It is not possible to draw any conclusions as to the elasticity of the vessel walls from a single time value

since that value depends on the length of the studied vessel. One can attempt to determine the length of the proximal aorta and compute the velocity, but exact measurement is made difficult by the inaccessible anatomical position of that segment.

We propose that the pulse wave propagation times t_1 and t for the proximal and distal segments of the aorta be placed in the relationship $\frac{t_1:t}{t_1}$ to one another, where one of the values t_1 represents unity. It could be postulated that in conditions diffusely affecting the elasticity of the aorta, the ratio of t_1 to t would remain identical with that observed under normal conditions, since any changes which occur affect both values equally. In atherosclerosis, which predominately damages the distal segment of the aorta, the ratio will change, mainly in the direction of diminution of the second value t, which refers to that segment.

Using this method, we studied 150 clinic patients, who were divided into three groups: the first group (34 persons) was made up of "healthy" individuals. These were students, staff members, and gastritis or cholecystitis patients who had been cleared by physical examination of any cardiovascular pathology. There were 24 men and 10 women from 12 to 74 years of age. The second group (84 persons) consisted of patients with clinical symptoms of cardiac atherosclerosis with hypertension. It contained 45 men and 39 women aged 30 to 83 years. The third group consisted of 32 patients with elevated arterial pressure due to chronic nephritis (13 persons) and hypertensive disease in the first and second stages without clear clinical symptoms of atherosclerosis. It contained 16 men and 16 women from 18 to 45 years old. We will not go into the clinical analysis of the materials, but will discuss only the results of piezoelectric pickup investigations.

Wave velocity in the "healthy" group ranged from 5 to 9.5 m/sec depending on age, which agrees with published data. The numerical values of t_1 and t are shown in the table, from which it can be seen that in healthy individuals most often t_1 = 0.04 to 0.07 sec and t = 0.06 to 0.08 sec. Thus, the ratio t_1:t was 1:1 to 1:2, i.e., either the values were identical, or t was greater than t_1.

Depending on age, the ratio t_1:t in the "healthy" group was as follows: under 20 years, 1:1.6; 21 to 30 years, 1:1.3; 31 to 40 years, 1:1.15; 41 to 50 years, 1:1.13; 51 to 60 years, 1:1.1; and 61 to 70 years, 1:1.0. The figures cited are averages based on measurements of t_1 and t in 5 to 7 persons from each age group.

The data cited show that with aging there occurs a gradual diminution of t which ends by its becoming equal to t_1. Such shifts can be explained by age changes in the aorta (N. N. Anichkov). In young persons, these changes and the elasticity of the aorta wall which they determine are more pronounced in the proximal segment, so that the value t_1, which corresponds to this segment, is less than t. With the years, age changes spread to affect the whole aorta, taking on a diffuse character, so that in addition to diminution of t_1, the value t begins to diminish as well; and an equalization of these two values takes place. Thus the "healthy" group as a whole was characterized by a ratio in which t was either greater than, or equal to, t_1.

In the group of atherosclerosis patients without hypertension, the velocity of the pulse wave (depending on the severity of the process) was from 9.5 to 25 m/sec. The numerical values of t_1 and t are shown in the table. In atherosclerosis usually t_1 = 0.05 to 0.08 sec, that is, almost identical to the healthy group, while t = 0.02 to 0.05 sec, which was noticeably less.

For the most part, t_1:t = 1:0.9 to 1:0.2, i.e., t was less than t_1. /81 These shifts in the ratio are explained by the way in which atherosclerotic changes are localized in the aorta. When they occur in the initial segments of the aorta, atherosclerotic changes do not attain a significant degree of development, and thus have little effect on the value t_1. As they spread distally they take on a more severe character in the thoracic, and still more so in the abdominal segments of the aorta, and in the iliac arteries--the segments for which t is measured. The latter value becomes less than t_1, with the degree of this diminution depending of the severity of the atherosclerotic changes in the lower parts of the aorta. The more fully they are developed, the shorter is the time t, and the greater the velocity V. Thus, the whole group of atherosclerotic patients was characterized by a ratio in which t was less than t_1.

In the group of patients with high arterial blood pressure without clear clinical symptoms of atherosclerosis, we were interested to observe the effect of elevated pressure on pulse wave velocity and on the ratio of t_1 and t. In this regard our study of the group of chronic nephritis patients was especially indicative, since in them hypertension is more rarely associated with atherosclerotic changes in the vessels (N. A. Ratner, G. L. Spivak). In half the patients of this group, despite the increased arterial blood pressure, V was normal (deviation from the mean norm did not exceed m ± 1 m/sec)[1]. The absolute values of t_1 and t were practically identical with those observed in the "healthy" group (see table), with t_1:t = 1:1 and higher.

In 16 patients, the velocity exceeded the mean norm by more than 1 m/sec, but nonetheless not by so great an amount that atherosclerosis could confidently

[1]As mean norm the velocity values given by V. P. Nikitin (1959) for various ages were used.

be diagnosed in them. Moderate increase in velocity may be due either to hypertension or to concealed atherosclerotic processes. These conditions must be differentiated. In 8 of the 16 patients, the ratio of t_1 to t was identical with that of the "healthy" group, permitting attribution of a certain increase in velocity to the effect of increased arterial pressure. In the remaining 8 patients the ratio of t_1 and t was similar to that observed in the atherosclerotic patients, that is, t was less than t_1 (1:0.8 to 1:0.6).

Thus, the majority of patients with elevated arterial pressure but with no clinical symptoms of atherosclerosis show essentially the same ratio of t_1 and t as in the "healthy" group. Shifts in the ratio in the direction of reduced values of the second index in some of them indicated a latent course of aortal atherosclerosis.

Conclusions.

1. Determination of pulse wave velocity from pulse curve lag behind EKG eliminates the need for the usual elaborate equipment, improves the quality of pulse curve recordings, and affords a basis for speculation as to the propagation time of the pulse wave in the proximal segment of the aorta.

2. Comparison of pulse wave propagation times in the proximal and distal segments of the aorta has revealed a characteristic pathognomic ratio specific to atherosclerosis. Correction for this ratio increases the specificity of the method for determining pulse wave propagation velocity in atherosclerosis.

3. Age changes have little effect, and functional changes (hypertension, hypertonus) have no effect on the ratio of t_1 and t because of the diffuse nature of these processes. This makes it possible to differentiate velocity increases in these conditions from velocity increase in atherosclerosis.

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